Early magnetic resonance imaging of brainstem lesions after severe head injury

RAIMUND FIRSCHING, PROF. DR. MED., DIETER WOISCHNECK, DR. MED., MICHAEL DIEDRICH, DR. MED., SUSAN KLEIN, M.D., ANDREAS RÜCKERT, M.D., HOLGER WITTING, DR. MED., AND WILFRIED DÖHRING, PROF. DR. MED.

Kliniks für Neurochirurgie und Diagnostische Radiologie, und Institut für Rechtsmedizin, Otto von Guericke Universität Magdeburg, Magdeburg, Germany

Object. The availability of magnetic resonance (MR) imaging data obtained in comatose patients after head injury is scarce, because MR imaging is somewhat cumbersome to perform in patients requiring ventilation and because, in the first hours after injury, its relevance is clearly inferior to computerized tomography (CT) scanning. The authors assessed the value of MR imaging in the early postinjury period.

Methods. In this prospective study MR imaging was performed in 61 consecutive patients within 7 days after they suffered a severe head injury. An initial CT scan had already been obtained. To understand the clinical significance of the lesions whose morphological appearance was identified with MR imaging, brainstem function was assessed by registration of somatosensory and auditory evoked potentials.

Brainstem lesions were visualized in 39 patients (64%). Bilateral pontine lesions proved to be 100% fatal and non-brainstem lesions carried a mortality rate of 9%.

In singular cases circumstances allowed for a clear clinical distinction between primary and secondary brainstem lesions. On MR imaging all lesions were hyper- and hypointense after intervals longer than 2 days. Within shorter intervals (<2 days) after the injury, primary lesions appeared isointense on MR imaging. In one secondary brainstem lesion there were no traces of blood.

Conclusions. Because mean intracranial pressure (ICP) levels in patients without brainstem lesions were similar to those in patients with brainstem lesions, the authors conclude that it was not mainly increased ICP that accounted for the high mortality rates in patients with brainstem lesions.

The authors also conclude that brainstem lesions are more frequently found in severe head injury than previously reported in studies based on neuropathological or CT scanning data. Early MR imaging after head injury has a higher predictive value than CT scanning.

Key Words • head injury • magnetic resonance imaging • brainstem injury

Our understanding of brainstem lesions after head injury has evolved with the improvement of imaging techniques. Before the era of computerized tomography (CT) scanning, primary focal brainstem lesions were considered to be a rare occurrence, never to be found isolated as a singular phenomenon.

Computerized tomography scanning is the procedure of choice to visualize intracerebral blood; unfortunately, the posterior fossa is usually hard to evaluate because of bone artifacts. Magnetic resonance (MR) imaging can disclose details of posterior fossa lesions, except for acute hemorrhages, up to 2 days postinjury. Because MR imaging is inferior to CT scanning in detecting acute hemorrhages, it has little practical value in the acute phase. In addition, special equipment is required to obtain MR images in a patient who is dependent on a respirator. Therefore, there is a paucity of MR imaging data obtained in comatose head-injured patients during the acute phase while the patient is dependent on the ventilator.

To study the role of primary and secondary brainstem lesions in a prospective study, we performed MR imaging of the head within 7 days of injury in 61 comatose patients who had suffered head injury. To corroborate the lesions identified with MR imaging, somatosensory evoked potentials (SEPs) and brainstem auditory evoked potentials (BAEPs) were also recorded and registered.

Clinical Material and Methods

In this prospective study, we studied MR images obtained in 61 consecutive patients admitted to the hospital in a comatose condition after suffering head injury. This study was approved by the local ethics committee. Severe head injury was defined as a head injury resulting in coma for at least 24 hours in spite of reducing sedation to the minimum required to ensure adequate respiration. Coma was defined as the state of consciousness in which patients did not open their eyes and did not obey com-
mands (Glasgow Coma Scale score of ≤ 7). All patients underwent initial cranial CT scanning on admission to the hospital. All patients were comatose at the time of the MR imaging studies. Patients with metal implants (such as fixators or pacemakers), those who had emerged from coma by the time MR imaging became available, or those who as a result of multiple injuries were too critically unstable for transfer to the MR imaging unit were excluded from this study. Patient ages ranged from 7 to 65 years (mean 23 years). The male/female ratio was 38:23.

Magnetic resonance imaging was performed within the first 7 days after head injury; the mean interval was 3.5 days. A 1.5-tesla magnet was used to obtain T₁- and T₂-weighted images. The midline and brainstem were investigated with T₂-weighted images in 3-mm sagittal plane slices. Finally, a coronal section was studied. Findings were classified as uni- or bilateral brainstem lesions according to their level or as nonbrainstem lesions. Furthermore, hypointense lesions were distinguished from hyperintense lesions.

In 41 patients SEPs and BAEPs were registered (techniques of registration have been reported elsewhere). Three types of findings were distinguished: bilateral reproducible potentials, unilateral reproducible potentials, and bilateral loss of evoked potentials. Results from the evoked potential monitoring were compared with the MR imaging findings.

Intracranial pressure (ICP) was recorded intraventricularly in 15 patients and epidurally in seven patients. The ICP levels (measured in millimeters of mercury) were compared with those measured during the clinical courses of patients with or without brainstem lesions. The ICP recordings were not made in the other patients in the group because of clinical circumstances (such as no lesion on the initial CT scan or admittance with wide fixed pupils) or because a large craniectomy or removal of the intracranial hematoma made ICP recording less urgent.

An extradural hematoma was removed in four patients and a subdural hematoma in 12 patients. A decompressive craniectomy was performed in eight patients, and in three patients an intracerebral space-occupying hematoma was removed.

Results

Magnetic Resonance Imaging Findings

Magnetic resonance imaging demonstrated no lesion in three patients, and 19 patients only had a supratentorial lesion. Brainstem lesions were identified in 39 (64%) of 61 patients (Fig. 1). Twenty-four of these 39 patients had a unilateral lesion of the brainstem (Table 1), cerebellum, or supratentorial lesions.

Seventeen (28%) of the 61 patients died. All 13 patients in whom a bilateral pontine lesion was found and four patients without a bilateral brainstem lesion died. The mortality rate for patients with a brainstem lesion, uni- or bilateral, disclosed by MR imaging was 44%.

Patients with brainstem lesions had other concomitant

<table>
<thead>
<tr>
<th>Location of Lesion</th>
<th>Extent of Lesion</th>
<th>No. of Patients</th>
<th>Patients W/ Fatal Outcome</th>
<th>Mortality Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>no brainstem lesion</td>
<td>—</td>
<td>22</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>mesencephalon</td>
<td>unilat or midline</td>
<td>12</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>pons or medulla oblongata</td>
<td>unilat</td>
<td>12</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>pons</td>
<td>bilat</td>
<td>13</td>
<td>13</td>
<td>100</td>
</tr>
<tr>
<td>lower portions of medulla oblongata</td>
<td>bilat</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* — = not applicable.
intracranial hemorrhages comparable to patients without brainstem lesions (Fig. 2).

### Evoked Potentials

The results of testing for evoked potentials are listed in Tables 2 and 3. Bilateral abolition of SEPs was noted in all cases of bilateral brainstem lesions. The SEPs were unilaterally preserved in all 10 patients who had a unilateral pontine or mesencephalic lesion.

In cases of bilateral pontine lesions, BAEPs were unilaterally preserved in three patients and abolished in seven. In 10 cases of unilateral mesencephalic or pontine lesions, the BAEPs were bilaterally preserved in six and unilaterally preserved in the other four.

### Intracranial Pressure

The difference between ICP levels in 14 patients with brainstem lesions and eight patients without brainstem lesions was not significant (Fig. 3). If a trend were to be distinguished, patients without a brainstem lesion tended to have higher ICP levels than patients with a brainstem lesion.

### Coma Duration

Survivors of brainstem lesions documented on MR imaging had a mean coma duration of 7 days, compared with 3.5 days in patients without brainstem lesions.

### Histopathological Findings

Postmortem findings of brainstem lesions in two cases verified by MR imaging disclosed perivascular hemorrhages, intraneuronal calcifications, and swollen axons. Differentiation of primary and secondary brainstem lesions by means of histopathological investigation was not possible.

### Unexpected Findings

**Case 1: Example of a Primary Brainstem Lesion.** This 22-year-old man was admitted to the hospital in a comatose state with wide fixed pupils 1 hour after being injured in an automobile accident. Computerized tomography scanning (Fig. 4 left) demonstrated marked traumatic subarachnoid hemorrhage but no focal lesion and no sign of increased ICP. To clarify the cause of the fixed pupils, an MR image (Fig. 4 right) was obtained during the 6th hour after the injury; the image disclosed fatal laceration of the pons. No other lesions appeared on CT or MR imaging in this case.

**Case 2: Example of a Secondary Brainstem Lesion.** This 12-year-old boy fell down a staircase at night and hit his head. At the time of the incident he was fully alert and returned to bed. A few hours later he was found comatose and arrived at the hospital with wide fixed pupils. In spite of immediate removal of the extradural hematoma, the boy did not recover and died 40 days later. Magnetic resonance images obtained on the 7th day postinjury disclosed ischemic lesions of the pons and cerebral peduncles with no traces of blood (Fig. 5).

### Discussion

**Incidence of Brainstem Lesions**

Neuropathological findings obtained during the pre–CT era suggested that traumatic brainstem lesions were an exceptional phenomenon that never occurred without concurrent diffuse brain damage.6 On cranial CT scanning brainstem lesions are difficult to detect because of bone artifacts within the lower portions of the posterior fossa. In 1993 Hashimoto, et al.,8 reported that 21 (8.8%) of 239 cases of severe head injuries exhibited evidence of brainstem lesions on cranial CT scanning. Five of seven of their patients with a pontine hemorrhage died. In the last few years MR imaging has been reported to detect intracranial lesions much more frequently; for example, lesions of the corpus callosum have been found in 22 to 49% of survivors of head injury.6,15

In this analysis the head injury was severe enough that all patients were comatose and dependent on a respirator (Glasgow Coma Scale score of ≤7) for a minimum of 24 hours. All patients were ventilated during the period in which MR imaging was performed. The frequency of brainstem lesion in this series of severe head injury cases was 64%. Mitchell and Adams10 questioned the existence of isolated brainstem lesions after head injury. In a postmortem study they found 18 of 100 patients had a primary brainstem lesion; these lesions were always associated with other concomitant lesions. In contrast to their conception of brainstem lesions, in the present study MR imaging performed in Case 1 confirmed the possibility of isolated brainstem lesions with no additional lesions, at
least visible on MR and CT images. Thus it appears that isolated primary contusion of the brainstem does occur; however, in most cases it is associated with concomitant lesions.

**Evoked Potentials**

One option that can be used to verify a lesion of the brainstem is registration of evoked potentials. The bilateral loss of cortical SEPs beyond 13 msec and BAEPs beyond Wave II is suggestive of a loss of brainstem function. The poor but accurate prognosis of this condition has been reported repeatedly. In this series, unilateral absence of SEPs coincided with unilateral pontine lesions on MR imaging in all 10 patients. In the 10 patients who had bilateral pontine lesions and in whom SEPs were registered, the abolition of SEPs was noted. In lesions of the mesencephalon this relation was less close.

Absence of BAEPs after head injury may well be due to petrous bone fractures. Their unilateral absence concurred in four of 10 patients with unilateral brainstem lesions on MR imaging; in the other six patients the BAEPs were still preserved bilaterally. Obviously lesions seen on MR im-

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**Fig. 3.** Bar graph showing the relationship among brainstem lesions and ICP and mortality rates.

**Fig. 4.** Case 1. Neuroimaging showing an example of a primary brain lesion. *Left:* Computerized tomography scan obtained in a 22-year-old man who initially presented with wide fixed pupils after being injured in a motor vehicle accident. Note the marked subarachnoid hemorrhage. *Right:* Magnetic resonance image obtained during the 6th hour after injury revealing hypo- and hyperintense lesions as an indicator of primary early traumatic bilateral pontine lesion (*arrows*). A ventricular catheter disclosed a mean ICP of 20 mm Hg; SEPs and BAEPs could not be elicited. Brain death was noted 4 days after the injury.
Magnetic resonance imaging after head injury

aging do not correlate well with loss of function in all cases. In three instances the BAEPs were preserved in spite of documented bilateral pontine lesions on MR imaging, clearly because the lesions had been ventral to the auditory pathway.

Thus, altogether brainstem function as assessed by registration of SEPs corroborated very well the morphological brainstem lesions disclosed by MR imaging.

**Magnetic Resonance Imaging Findings**

In all but two brainstem lesions (Cases 1 and 2), MR imaging exhibited mixed hyperintense and hypointense signals within the lesion. Magnetic resonance imaging performed within 6 hours of injury in Case 1 showed a pontine laceration, but the tissue was isointense on T1- and T2-weighted images. It obviously was too early to demonstrate breakdown metabolites of blood with hyperintense signals, but destruction of the nervous tissue could be discerned. Because the ICP was normal and CT and MR imaging demonstrated no signs of increased ICP 6 hours postinjury, the brainstem lesion was concluded to be caused by the primary impact rather than by a secondary brainstem compression.

In Case 2 the brainstem lesion undoubtedly had a secondary origin; it was caused by herniation from the extradural hematoma and not by the impact itself. This was the only patient who had a secondary lesion and the only patient with hypointense and hyperintense signals of the pons and cerebral peduncles on both T1- and T2-weighted images, denoting edema but no traces of blood on the 7th day after the injury. Because the patient initially had exhibited no neurological disorders after the injury, his brainstem must have been intact. Apparently herniation from the extradural hematoma caused a slowly increasing compression mainly of the peduncles but did not lead to hemorrhage in spite of its fatal effect. Because it is very rare to encounter a purely secondary brainstem lesion, there are no MR imaging data on this observation available to us in the literature. Bernardi, et al.,1 support the idea that these lesions must be hemorrhagic but do not supply an example. Thus, possibly, signal intensity may serve to distinguish primary lesions caused by direct destruction of nervous tissue either as a laceration or as a shear lesion from secondary brainstem lesions caused by herniation.

Aside from lesions disclosed by MR imaging it must be kept in mind that there are most likely lesions that are not depicted on MR imaging. At 6 months postinjury single-photon emission CT scanning has been reported to demonstrate lesions that escaped detection on MR imaging.11

**Intracranial Pressure**

If a primary brainstem lesion occurred at the time of impact and a secondary lesion was caused by brainstem compression, the latter could be expected to present with increased ICP at some point after the injury. Intracranial pressure levels in eight patients with nonbrainstem lesions with a mortality rate of 13% (one of eight) were of the same order as ICP levels in five patients with bilateral brainstem lesions (Fig. 3). Because the mortality rate for patients with brainstem lesions was much higher than that for patients with nonbrainstem lesions, it appears that the brainstem lesion itself is the cause of death rather than the increase in ICP.

![Fig. 5. Case 2. Magnetic resonance imaging showing an example of a secondary brain lesion obtained in a 12-year-old boy with secondary ischemic brainstem lesions after herniation from an extradural hematoma and subsequent surgical removal and decompressive craniotomy. The MR imaging findings indicate edema but no traces of blood (arrows).](image)

This notion may be supported by the fact that in all patients who had a bilateral brainstem lesion and a fatal outcome, coma invariably persisted from the time of hospital admission to death. None of these patients regained consciousness for even a short time. It appears more likely that the brainstem lesions were acquired primarily at the time of the original impact and not secondarily as a result of the gradually increasing ICP. There were no “talk-and-die” patients in the group with brainstem lesions. In future studies the relationship between MR imaging findings and ICP levels should be investigated in larger numbers of patients to understand more clearly the significance and frequency of brainstem compression compared with primary laceration.

**Prognosis of Brainstem Injury**

Quality of life has been reported to be related to the depth of the lesion identified on MR imaging in surviving patients.3 Magnetic resonance findings have also shown a relationship with posttraumatic amnesia and coma duration.14 In this series, duration of coma in survivors with brainstem lesions was twice as long as the duration of coma in survivors without brainstem lesions despite similar ICP levels.

A bilateral pontine lesion observed on MR imaging in this series proved to be fatal without exception. Two of 24 patients with a unilateral brainstem lesion died of pulmonary complications 2 weeks and 2 months, respectively, after regaining consciousness and being fully mobilized. Bilateral pontine lesions observed on MR imaging have a high predictive value; in their presence the mortality rate was 100% and in their absence it was 8%. A practical aspect of this diagnostic tool may be that further surgery such as decompressive craniectomy should be
warranted only when there is a chance of survival in the absence of bilateral pontine lesions.

Conclusions

This analysis of early MR imaging in 61 comatose patients after severe head injury disclosed an incidence of brainstem lesions of 64%. The patient’s medical history and clinical findings were suggestive of primary brainstem lesions suffered at the time of impact in 38 of 39 patients. This incidence is much higher than findings from earlier series based on CT scanning or neuropathological data. Because these brainstem lesions, which were undetected by CT scanning, apparently have great predictive value, early MR imaging should be required for future studies on the effect of drugs or different regimens of treatment in head injury to ensure that subgroups are comparable.

The MR imaging findings also suggest that primary brainstem lesions may be distinguished from secondary brainstem lesions by their signal intensity.

References


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Address reprint requests to: Raimund Firsching, Prof. Dr. med., Klinik für Neurochirurgie, Otto von Guericke Universität Magdeburg, Leipziger Straße 44, 39120 Magdeburg, Germany.